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REVIEW

Minimally invasive treatment of pancreatic pseudocysts

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Abstract

A pancreatic pseudocyst (PPC) is typically a complication of acute and chronic pancreatitis, trauma or pancreatic duct obstruction. The diagnosis of PPC can be made if an acute fluid collection persists for 4 to 6 wk and is enveloped by a distinct wall. Most PPCs regress spontaneously and require no treatment, whereas some may persist and progress until complications occur. The decision whether to treat a patient who has a PPC, as well as when and with what treatment modalities, is a difficult one. PPCs can be treated with a variety of methods: percutaneous catheter drainage (PCD), endoscopic transpapillary or transmural drainage, laparoscopic surgery, or open pseudocystoenterostomy. The recent trend in the management of symptomatic PPC has moved toward less invasive approaches such as endoscopic- and image-guided PCD. The endoscopic approach is suitable because most PPCs lie adjacent to the stomach. The major advantage of the endoscopic approach is that it creates a permanent pseudocysto-gastric track with no spillage of pancreatic enzymes. However, given the drainage problems, the monitoring, catheter manipulation and the analysis of cystic content are very difficult or impossible to perform endoscopically, unlike in the PCD approach. Several conditions must be met to achieve the complete obliteration of the cyst cavity. Pancreatic duct anatomy is an important factor in the prognosis of the treatment outcome, and the recovery of disrupted pancreatic ducts is the main prognostic factor for successful treatment of PPC, regardless of the treatment method used. In this article, we review and evaluate the minimally invasive approaches in the management of PPCs.

Key words: Complications; Pseudocyst; Treatment; Drainage; Outcomes

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Core tip: Pancreatic pseudocysts (PPCs) are common complications of acute and chronic pancreatitis, pancreatic trauma, and pancreatic duct obstruction. They can be treated with a variety of methods: percutaneous catheter drainage, endoscopic transpapillary or transmural drainage, laparoscopic surgery, or open pseudocystoenterostomy. It is a difficult decision whether to treat a patient with a PPC and if so, with what treatment modalities and when. This article presents and critically evaluates the minimally invasive approaches for the treatment of PPCs.

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INTRODUCTION

A pancreatic pseudocyst (PPC) is defined as a collection of fluid in the peripancreatic or intra-pancreatic tissues, is surrounded by a well-defined wall and contains essentially no solid material^[1]. PPCs are usually complications of both acute and chronic pancreatitis, pancreatic trauma, and pancreatic duct (PD) obstruction^[2-6].

It is a difficult decision whether to treat a patient who has a PPC, and if so, when and with what treatment modalities. PPCs should initially be managed conservatively because many resolve spontaneously within 4 to 6 wk. Although most PPCs regress spontaneously and require no treatment, some (especially those larger than 6 cm) require treatment to prevent cystic infection, rupture, haemorrhage, and the resultant obstruction of the stomach, small bowel, colon or bile ducts^[7,8].

Traditionally, surgical approach was the treatment of choice for symptomatic PPCs. Although surgery is effective, complications can occur in up to 35% of patients, and death from surgery has also been noted^[9]. The recent trend in the management of symptomatic PPC has been toward less invasive approaches such as endoscopic and image-guided percutaneous catheter drainage (PCD)^[2-13].

Several conditions must be met to achieve the complete obliteration of the cyst cavity. PD anatomy is an important factor for the prognosis and the treatment outcomes. When PPC-PD communication is identified, the mean duration of drainage increases to weeks or months, depending on the condition of the PD. The recovery of a disrupted PD is the main prognostic factor for successful treatment of PPC regardless of the treatment method^[2,11,12,14]. This

review article presents and critically evaluates the minimally invasive approaches for the treatment of PPCs.

DIAGNOSIS OF PPCS

The distinction between PPC and other similar entities, such as benign and malignant cystic lesions, vascular pathology such as pseudoaneurysms and hematomas, seromas, abscesses, and bilomas, is crucial in the decision to treat a patient who has a PPC, as well as when and by which method. This requires the correlation of often complex and overlapping clinical presentations and laboratory findings with those of imaging studies, such as ultrasound (US), computed tomography (CT), and magnetic resonance imaging (MRI)^[15,16].

Clinical presentation and laboratory findings

PPC is typically asymptomatic, and its clinical presentation tends to occur in cases with complications throughout their clinical course. During physical examination, the most common presenting symptoms that might be attributed to the development of symptomatic PPC are persistent abdominal pain and/or an epigastric mass with a persistently raised serum amylase level^[17]. Clinical presentations of PPC complications are infection, rupture and haemorrhage^[7,17]. Infection occurs in approximately 10% of cases and is characterized by fever and abdominal pain. The leakage of the content from the PPC into the peritoneum can cause the appearance of pancreatic ascites. However, sudden rupture of the PPC into the peritoneum produces severe peritonitis that is often fatal. Haemorrhage is caused by the erosion of the small vessels that line the cyst wall or the erosion of surrounding major blood vessels. Intracystic bleeding leads to a rapid enlargement of the PPC, which produces pain and shock. Spontaneous rupture of the PPC into the gastrointestinal tract can result in the drainage of its contents into the gastrointestinal tract and the amelioration of symptoms. However, this is often associated with vomiting, hematemesis and melena^[17].

Laboratory findings have a limited value in the diagnosis of PPC. Serum amylase and lipase levels are persistently elevated in up to 76% of patients with PPC. When PPC produces a biliary obstruction, the serum bilirubin level is increased^[17].

Imaging evaluation

The diagnosis of a PPC is usually established by imaging studies, whereby a rapid progress in the improvement of diagnostic modalities enables detection with high sensitivity and specificity.

Because transabdominal ultrasonography is a very inexpensive and noninvasive technique, it should be performed as a first step in the diagnosis of PPCs.

US has a diagnostic sensitivity of 75% to 90% in detecting PPCs and the technique is highly dependent on the experience of the examiner. It has a limited role in the assessment of small PPCs (smaller than 10 mm). However, small PPCs are asymptomatic and without clinical significance, usually not requiring any treatment^[2,17].

Endoscopic ultrasound (EUS) may typically display a small PPC, being the best method in distinguishing acute fluid collections from pancreatic abscesses and PPCs, with high sensitivity (93% to 100%) and specificity (92% to 98%)^[18,19]. Besides, EUS can accurately define the proximity of the PPC to the gut lumen and surrounding large blood vessels. Limitations of EUS are its inability to demonstrate large PPCs which extend into peripancreatic areas in their entirety, and display PPCs which are more than 1 cm distant away from the gastric or duodenal wall^[20,21].

CT scanning is a standard and precise imaging modality in the setting of PPCs, with 82% to 100% sensitivity and 98% specificity. CT scan is more effective than US in detecting the secondary complications of a PPC, such as infection; hemorrhage, and involvement of adjacent structures^[18,22].

Endoscopic retrograde cholangiopancreatography (ERCP) may be useful in patients who require delineation of PD anatomy, helping to devise optimal therapy. Although ERCP provides less information regarding the pancreatic size and surrounding visceral structures than CT and ultrasound, it renders important information on the anatomy of the pancreatic and biliary ductal system^[14].

Magnetic resonance imaging (MRI) is a good alternative to CT for detection of PPCs due to its ability to characterize pancreatic and peripancreatic collections as partially or fully fluid in consistency. Magnetic resonance cholangiopancreatography (MRCP) may replace ERCP in the diagnostic evaluation of pancreatic duct. However, the diagnosis of PPC-PD communication is rather difficult because a communication can only be identified by MRCP if a high-intensity fluid tract can be detected between the pseudocyst and the duct^[2,21,23-26].

A plain radiograph of the abdomen is rarely helpful in diagnosing PPC. Occasionally, it may demonstrate displacement of the gastric bubble or calcification in the cyst wall. A chest radiograph may show elevations of the diaphragm, pleural effusion, or a mediastinal mass.

Differential diagnosis between pseudocysts and cystic neoplasms

The differential diagnosis between PPCs and cystic neoplasms may be difficult in patients with a pancreatic fluid collection. Clinical criteria such as prior episodes of acute pancreatitis, and data regarding chronic pancreatitis or a calcified cystic wall less than 1 cm thick, make the diagnosis of PPC more likely. On the contrary, weight loss, a palpable abdominal

mass, the lack of pre-existing pancreatic disease, and multilocular cysts with non-calcified walls thicker than 1 cm, all indicate the likelihood of a cystic malignant tumour. EUS or US-guided diagnostic puncture and sampling of the fluid content and of the PPC wall helps to distinguish cystic malignancies from PPCs^[27-29]. Research has recently focused on the identification of new biomarkers for the diagnosis of malignant lesions. Important criteria for malignancy are a markedly elevated carcinoembryonic antigen (CEA) value in the cyst fluid (over 192 ng/mL) and increasing viscosity of the cyst content^[18,30].

INDICATIONS FOR TREATMENT OF PPCS

The most important question in clinical practice related to acute or chronic PPCs is whether and when they should be treated. A careful preliminary clinical and imaging evaluation of benign pancreatic fluid collections can avoid unnecessary interventions. The majority of the simple PPCs are asymptomatic and do not require interventional treatment. Treatment is indicated if the complications are present or whether intervention is necessary to prevent complications. The indications for interventional procedures in the treatment of PPCs are summarized in Table 1.

Symptoms result from biliary obstruction, the effects of painful or obstructive masses, infection or haemorrhage into the cyst, pancreaticopleural fistula or compression of the surrounding major vessels, and in such cases, interventional treatment is typically indicated. Treatment is also indicated for symptomatic PPCs that cause abdominal distension, nausea and vomiting, pain, or gastrointestinal bleeding (Table 1).

PPCs larger than 4 cm that develop outside the pancreas can be considered independent predictive factors of persistent symptoms because they rarely regress spontaneously and can cause complications^[31]. Therefore, if they demonstrate either unchanged size and morphology or progression over a period of more than 6 wk of observation, these are relative indications for treatment^[15,31]. A relative indication for treatment includes PPCs whose wall thickness is between 5 and 10 mm and PPCs caused by the presence of chronic pancreatitis with duct abnormalities or stones in the PD. In these patients, constant irritation promotes inflammation and reduces the rate of spontaneous regression^[18,31,32]. Whenever a malignant tumour is suspected, surgical treatment is urgently indicated (Table 1)^[33,34]. When an intervention is required, the best option should be the application of a multidisciplinary approach based on the initial imaging and clinical findings.

MINIMALLY INVASIVE APPROACHES TO THE MANAGEMENT OF PPCS

PPCs as benign fluid collections in the pancreas can

Table 1 Indications for therapeutic intervention for pancreatic pseudocysts

Clinical presentations and complications
<i>Local complications</i>
Infection of pancreatic pseudocyst
Hemorrhage into pancreatic pseudocyst
Rupture (can cause pancreatic ascites, shock and peritonitis)
<i>Involving adjacent organs</i>
Gastrointestinal tract:
Esophagus (secondary achalasia, mechanical dysphagia)
Stomach (clinically relevant gastric outlet stenosis, fistula, intramural gastric mass)
Duodenum (clinically relevant duodenal stenosis, fistula)
Colon (clinically relevant colonic stenosis and/or rectal bleeding)
Liver (stenosis of the common bile duct with jaundice due to compression)
Vascular:
Arterial (erosion of gastroduodenal and/or splenic artery)
Venous (thrombosis of portal and/or splenic vein)
Spleen (splenic rupture)
Genitourinary tract (stricture, fistula, ureter obstruction)
Chest (pancreaticopleural fistula, pleural effusion, mediastinal extension)
Skin (subcutaneous fat necrosis)
<i>Symptomatic pancreatic pseudocyst</i>
Abdominal distension
Nausea and vomiting
Pain
Upper gastrointestinal bleeding
<i>Relative indications for intervention in asymptomatic pancreatic pseudocyst</i>
Pseudocyst > 5 cm, unchanged in size and morphology for more than 6 wk ^[15]
Pseudocyst > 4 cm and extrapancreatic complications in patients with chronic alcoholic pancreatitis ^[31]
Cyst wall > 5 mm (mature cyst) ^[32]
Chronic pancreatitis with advanced pancreatic duct changes ^[31]
Suspected cystic pancreatic tumor (requiring surgery) ^[33,34]

mimic cystic neoplasms. Therefore, pretreatment evaluations of pancreatic fluid collections for appropriate therapeutic intervention should be focused on the exclusion of cystic neoplasms that masquerade as pseudocysts^[35]. The topic of cystic neoplasms of the pancreas is broad, and thus this article focuses primarily on the minimally invasive treatments of benign PPCs. Once a PPC has been diagnosed, it must be determined whether it can be treated conservatively in hopes of a spontaneous resolution, or whether an intervention is necessary to prevent complications. If an intervention is necessary, it must be determined whether surgical, PCD, or endoscopic drainage (ED) is the best approach.

Conservative management

Based on earlier studies on the clinical course of PPCs, the rate of spontaneous resolution of PPCs has been reported to be from 8% to 70%^[15,17,18]. This wide range can be attributed to many factors that influence PPCs, including size, chronicity, wall thickness, multiplicity, and aetiology.

The size of the PPC is an important determinant of

spontaneous resolution. The majority of pseudocysts that are over 6 cm in size that persist for over 6 wk have been regarded as unlikely to resolve spontaneously^[17,18,31,36]. However, some large PPCs (> 6 cm) may undergo spontaneous resolution, which suggests that the size of the PPC alone is not an indication for drainage^[36,37].

Chronicity adversely affects the healing of PPCs whereby PPCs that persist for 8 to 10 wk are unlikely to resolve spontaneously. Most PPCs that are likely to heal do so within 6 wk, but the resolution may occur after 24 wk or even 28 mo^[17]. Chronic pancreatitis and pancreatic calcifications are also poor prognostic indicators^[18,38].

Other factors that indicate that spontaneous regression is less likely include the presence of multiple cysts^[18,39], location in the tail of the pancreas^[37], and a wall thickness greater than 1 cm^[40]. The aetiology may also have some bearing on the outcome. PPCs related to alcohol abuse have a more favourable outcome compared with those of biliary aetiology. However, traumatic PPCs may have a high percentage of spontaneous resolution^[39].

The setting of non-interventional conservative management of PPCs is still poorly evaluated. Earlier studies showed that conservative treatment in the hope of spontaneous resolution was not without risks. Several studies have warned against serious, life-threatening complications related to the conservative treatment of PPCs^[41-44]. However, with improved medical care, the incidence of complications as well as the mortality rate has decreased considerably. Several studies^[36,41,45,46] have reported that some patients with PPCs can be managed conservatively if the presenting symptoms can be controlled. According to their results, the complication rates with conservative management are low (< 1%).

These results suggest that some patients with PPCs can be managed conservatively and that some pseudocysts can resolve with supportive medical care. Medical care includes the use of intravenous fluids, analgesics and antiemetics to control the presenting symptoms caused by PPC. For patients who can tolerate oral intake, a low fat diet is recommended, whereas for those who cannot tolerate oral nutrition, support can be provided *via* nasoenteral feeding or total parenteral nutrition^[47].

Somatostatin (octreotide) has an inhibitory effect on pancreatic exocrine secretion, and it can be used to decrease the pancreatic secretion, which leads to the resolution of PPC. Octreotide has also been used in conjunction with PCD of PPCs, which results in a shorter drainage time. The role of somatostatin in the management of PPCs is not clear because this treatment has not been adequately tested and only a handful of case series have been published^[47-49]. Prospective controlled trials are necessary to demonstrate its efficacy.

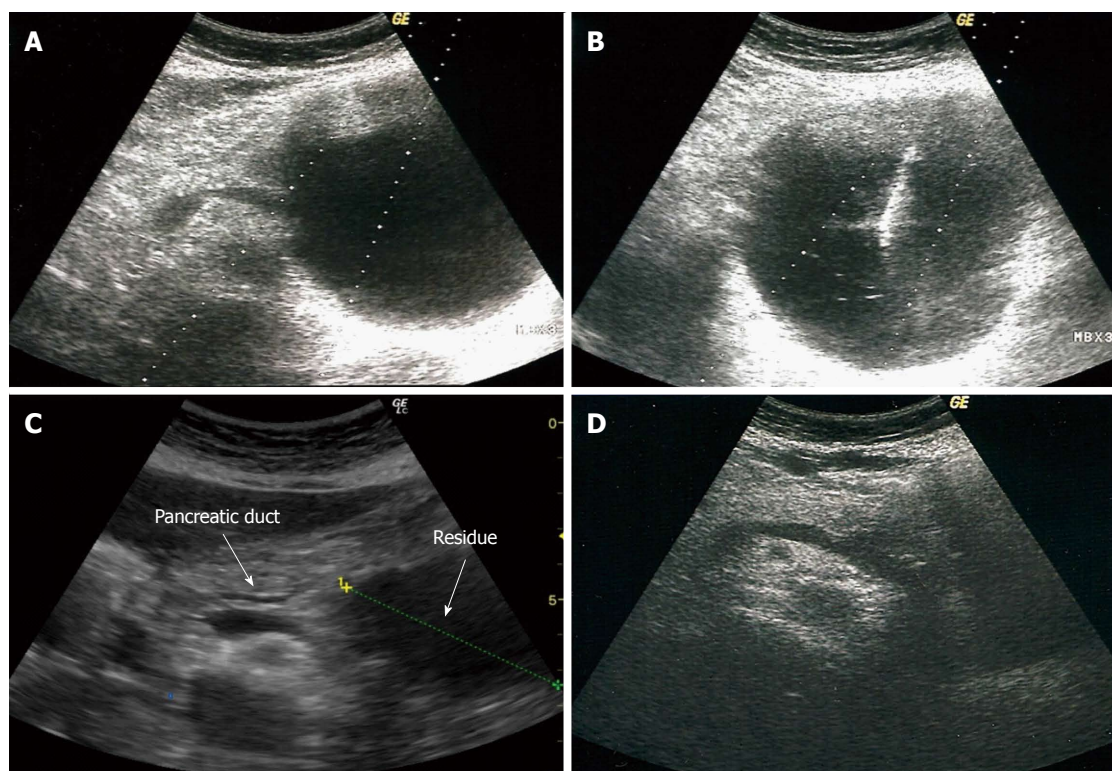


Figure 1 Appearance on ultrasound of a pancreatic pseudocyst before, during and after treatment. A: Appearance on ultrasound of a PPC in the tail of pancreas before treatment; B: Insertion of a catheter into the PPC; C: Residue of PPC with suspected PPC-PD communication (marked by arrows) immediately after the procedure; D: The appearance of the pancreas several months after the procedure. PPC: Pancreatic pseudocyst; PD: Pancreatic duct.

Image-guided percutaneous treatment of PPCs

Image-guided percutaneous drainage of PPCs is a well-established and relatively inexpensive drainage method that involves either simple percutaneous aspiration or PCD. It is most commonly performed under ultrasound or CT control, and in some cases, under MRI or fluoroscopic guidance (Figure 1)^[2,4,8,10,17,21,35,47,50-52].

Single-step needle aspiration of PPCs is associated with a high recurrence rate (70% or more) and cannot be considered the optimal treatment^[4,8]. The continuous vacuum drainage system is more effective because it continuously evacuates the cyst content and thereby avoids the lytic action of pancreatic enzymes that may lead to obliteration of the cyst cavity. This approach has achieved high initial drainage success rates (70%-100%) and reduced recurrence rates^[4,8,23,53].

Several conditions must be met to achieve the complete obliteration of the cyst cavity. PD disruption is the initial pathologic event that triggers PPC formation, and its anatomy is an important factor in the prognosis of the complete obliteration of the cyst cavity. Therefore, the complete removal of liquid and air, which is necessary to keep the cyst walls in close contact, constitutes the mechanical aspect of obliteration. The recovery of a disrupted PD has been recognized as the main prognostic factor for successful treatment of PPC regardless of the treatment method used^[8,10,14]. Patients with PPC-PD communication

require a longer duration of drainage, as short-term drainage results in very high recurrence rates. However, some authors consider that the risk of septic complications is potentially increased with prolonged drainage periods^[2,8,14,53-55].

Percutaneous techniques are usually performed under local anaesthesia and seem technically feasible in the vast majority of patients with PPCs. Transperitoneal, retroperitoneal, transhepatic, transgastric, and transduodenal approaches are typically used. The access route for drainage depends on the size, location, and the disposition of the surrounding viscera and blood vessels^[2,4,8,23,51]. Depending on the operator's experience, the tandem trocar technique or the Seldinger technique may be used. If the Seldinger technique is used, the catheter tract should be sequentially dilated over a guidewire. The use of three-dimensional ultrasound and colour Doppler may help to guide the catheters into the cyst cavities and aid in the circumvention of major vascular structures, which increases the safety of the procedure^[2,4,17,21,47,52].

After complete evacuation of the cystic content, the catheter should be secured to the skin and connected to a pressure bag for continuous external drainage. Catheter exchange may be performed as indicated. When the PPC has resolved and the drainage output becomes minimal (less than 10 mL/d), the catheter should be removed^[8]. Percutaneous drainage is a safe and effective method for treatment of PPCs.

Complications include catheter-related secondary infections (9%), bleeding (1%-2%), inadvertent traversing of the pleural space or other viscera (1%-2%), catheter occlusion, cellulitis at the site of entry, and sepsis^[4,55]. Another limitation of PCD is the development of pancreaticocutaneous fistulae. However, the resulting pancreatic-cutaneous fistula spontaneously resolves with time in 60% to 70% of patients^[4,56]. Moreover, in some cases, the fistula can be successfully treated by image-guided PCD^[57]. In the case of superinfection or drainage problems, monitoring, catheter manipulation and analysis of the cystic content can be performed much more easily by PCD than by an endoscopic approach^[8,21,58,59].

Endoscopic drainage of PPCs

ED provides minimally invasive access to the PPC, which may be performed by a trans-papillary or a trans-mural approach. Sometimes a combination of both methods may be necessary to drain a pseudocyst. ED is suitable because most PPCs lie adjacent to the stomach; however, both endoscopic and radiologic skills are required here. The aim of endoscopic treatment is to create a connection between the pseudocyst cavity and the gastrointestinal lumen^[60].

Transpapillary/transductal endoscopic drainage is recommended for PPCs that communicate with the main PD or one of its side branches located in the head or the body of the pancreas. A limited number of PPCs may be drained *via* a transpapillary insertion of a stent that bridges the main pancreatic duct or a disrupted side branch. A favourable predictor of successful therapy is a dilated Wirsung duct above a stenotic area underneath the stent^[4,7,61,62].

This technique involves pancreatic endoscopic sphincterotomy, balloon dilatation of the commonly detected PD strictures, and insertion of a guidewire through the duct directly into the pseudocyst cavity. Thereafter, a plastic stent of 5 F to 7 F (up to 10 F) in diameter is inserted over the wire^[4,23,63-65]. The duration of stenting depends on the clinical course of PPC regression^[23,61]. Stents should be left in place for a longer duration because their removal within 2 mo is associated with a higher incidence of pseudocyst recurrence^[66]. Some authors have reported on the routine exchange of stents every 6 to 8 wk for as long as the PPCs remained unresolved^[64].

Transpapillary drainage appears to be a safe and effective procedure (the immediate success rate is approximately 85%) with low morbidity (6%) and no reported mortality. The best results are obtained when the pseudocyst is older than 6 mo or smaller than 60 mm^[64,67]. Haemorrhagic complications occurred in less than 1% of patients and pancreatitis occurred in 5%. Stent clogging, which can lead to infection, can be treated with stent changes alone. Broad-spectrum antibiotics are administered in cases of infected PPCs^[4,23,61,64,65].

Transmural endoscopic drainage (cystogastrostomy or cystoduodenostomy) is indicated for pseudocysts that do not communicate with the main PD and that are compressed against the digestive tract. Drainage of the cyst fluid by the trans-mural approach is achieved *via* the insertion of a stent between the pseudocyst and the gastric lumen (cystogastrostomy) or between the pseudocyst and the duodenal lumen (cystoduodenostomy). The drainage procedure may be performed either by direct endoscopy as a "semi-blind" procedure if an impression caused by the cyst is present, or by EUS guidance. Technically, cystoduodenostomy should be given preference over cystogastrostomy if both routes are deemed equally feasible. Direct endoscopic drainage can be performed only if the PPC is located next to the gastric or the duodenal lumen. The site of transmural puncture for a direct endoscopic intervention should be determined visually and fluoroscopically by an observed bulge that represents the extrinsic compression of the collection into the gut lumen^[2,68-71]. Once the bulge is located, its apex can be identified as the optimal needle insertion site. After needle puncture and aspiration of the pseudocyst content (for biochemical and cytological analyses), a guidewire should be inserted along which an incision can be made with either a diathermic coagulation probe or a needle-knife papillotome. Once access has been achieved, a double pigtail catheter can be introduced into the cyst over the wire. The European Society of Gastrointestinal Endoscopy (ESGE) recommends the insertion of at least two double-pigtail plastic stents. Transmural stents should not be retrieved before complete resolution of the PPC as determined by cross-sectional imaging, and not before 2 mo of stenting^[23,65-67,72-74].

However, a bulge is often absent with smaller collections, collections with low serum albumin, and collections in or near the pancreatic tail. Therefore, to minimize the risk of complications such as the puncture of adjacent structures, bleeding, and perforation, EUS is increasingly used to perform ED^[2,75]. Randomized clinical trials of endoscopic transmural drainage with and without EUS guidance showed that EUS-visualization had an advantage over conventional ED^[68,69]. Even in large bulging pseudocysts, the EUS-guided drainage is superior to the purely endoscopic approach because the puncture of vascular structures and bleeding into the collection can be avoided during and immediately after the procedure by Doppler sonographic visualization^[2,20,76]. The use of EUS-guided drainage has been reported, especially for PPCs that do not bulge onto the gut wall or PPCs with parietal vessels due to portal hypertension^[4,32,64,77]. The stent type used for endoscopic drainage is currently a major area of interest. Conventional drainage with plastic stents has its limitations. A covered self-expandable metallic stent is an alternative to conventional drainage with plastic stents because it offers the option of access

to the fistula *via* a larger diameter for drainage, which may increase the final success rate. One problem with covered self expandable metallic stent is dislodgement, so a metallic stent with flared or looped ends at both extremities may be the best option^[78-80].

The advantages of the endoscopic approach compared with PCD include internal drainage and avoidance of external fistulae, but limitations include the need for multiple repeated procedures under sedation or anaesthesia; it is also necessary that the location of the PPC be further than 1-1.5 cm from the gut wall^[20,65,67,81]. Moreover, in the case of superinfection or drainage problems, the monitoring, catheter manipulation and the analysis of cystic content are very difficult by ED^[8,11,21,82]. A combination of a percutaneous approach with endoscopic transmural drainage can prevent external fistulae and avoid repetitive endoscopic interventions^[83].

Some authors advocate the use of a combination of transmural and transpapillary techniques to drain pseudocysts. They have used ERCP in the same endoscopic session to assess and treat any PD leakage; when PD leaks are evident, ERCP is also used for the placement of PD stents to bridge the leak site or stricture. Thus, when the treatment of the cause of the pseudocyst (*i.e.*, the duct leak) is possible by placement of concomitant PD stents, this has been shown to yield better outcomes^[13,61,81,84]. Additionally, in patients with disconnected duct syndrome, transgastric stent removal results in a lack of a conduit for drainage of pancreatic secretions, which leads to pseudocyst recurrence^[13,85].

Laparoscopic surgery

The classic open surgical approach for the treatment of PPC requires a laparotomy with attendant risks of morbidity and mortality. The development of advanced laparoscopic techniques and technologies offer new modalities for the treatment of PPCs. Laparoscopic surgery is a method in which the lumen of the PPC is anastomosed either to the posterior stomach wall or to the jejunum with a linear endoscopic stapler or with laparoscopic suturing techniques; this provides ongoing internal drainage and decompression of the PPC^[4,7,23,86,87].

Laparoscopic drainage of mature PPCs is usually the definitive treatment because it is associated with a low complication rate and a good outcome in the postoperative follow-up period. Currently, most PPCs can be approached and managed by a laparoscopic approach, which is due to the availability of advanced imaging systems and cameras, better haemostatic equipment and excellent suturing skills^[23,88]. Laparoscopic procedures for PPC include pancreatic pseudocystogastrostomy, pseudocystoduodenostomy, and pseudocystojejunostomy.

Cystogastrostomy is the most commonly used laparoscopic procedure, and it can be performed *via*

the endogastric, transgastric, or exogastric routes. In cases where pseudocysts contain significant debris because of the larger size of the stoma that is created, laparoscopy seems to have a distinct advantage over endoscopic drainage^[4,7,23].

Several authors reported that laparoscopic drainage was associated with low morbidity (early postprocedure bleeding and infection), rapid recovery, and recurrence rates comparable to those reported for open surgery. The disadvantage of laparoscopic surgery is that it may not be suitable for patients who are unfit to undergo general anaesthesia or for patients who had undergone extensive previous abdominal surgery. Although laparoscopic management has been reported with encouraging results, long-term follow-up results have yet to show equivalence to those of open surgery. Additionally, randomized controlled trials that compare PCD, laparoscopic and ED techniques for the management of PPCs are required^[7,8,23,89].

CONCLUSION

PPC usually runs asymptotically and its clinical presentation mainly occurs in case of complications during its clinical course. Once a PPC is diagnosed, it must be determined whether it can be treated conservatively with the hope of spontaneous resolution, or if an intervention is necessary to prevent complications. The setting of conservative management of PPCs is still poorly evaluated. Several studies have reported that some patients with PPCs can be managed conservatively with supportive medical care if the presenting symptoms can be controlled.

If intervention is necessary, it must be determined whether surgical treatment, PCD, or ED is the best approach. Much overlap exists in the various treatment options offered by interventional radiologists, gastroenterologists, and surgeons, and often a combined approach is needed. Several conditions must be met to achieve the complete obliteration of the cyst cavity. PD anatomy is an important factor in the results of the treatment. When PPC-PD communication is identified, the mean duration of drainage increases to weeks or months, depending on the condition of the PD. The recovery of disrupted PD is the main prognostic factor for successful treatment of PPC regardless of the treatment method used.

Traditionally, symptomatic PPC has been treated by surgical internal drainage. However, this treatment involves considerable surgical trauma and general anaesthesia.

The recent trend in the management of symptomatic PPC has moved toward less invasive approaches such as ED, image-guided PCD and minimal invasive surgery.

ED of PPCs may be performed by a trans-papillary or a trans-mural approach and is suitable because most PPCs lie adjacent to the stomach. The major

advantage of the endoscopic approach is that it creates a permanent pseudocysto-gastric track with no spillage of pancreatic enzymes, which reduces the risks of formation of pancreatocutaneous fistulas; this is in contrast to PCD. Moreover, PCD that persists too long is not practical, especially for young and professionally active patients. Therefore, some authors suggest that ED should be the preferred modality for PPCs that are located immediately adjacent to the gastric or duodenal wall.

However, with these potential drainage problems (which could appear with both techniques), the monitoring, manipulation or change of stent, and the analysis of cystic content are very difficult or impossible to perform endoscopically, unlike the PCD approach. Moreover, PCD is less aggressive compared with surgical and endoscopic (especially with ERCP) methods, is suitable for the treatment of all PPCs regardless of their location and can be performed without general anaesthesia. Therefore, this treatment option is especially recommended for patients who are unsuitable for more aggressive methods and for those at a high risk for complications of general anaesthesia.

Some authors advocate the use of a combination of transmural and transpapillary techniques to drain pseudocysts. Some have used ERCP in the same endoscopic session to assess and treat any PD leakage, and when PD leaks were evident, ERCP was used for the placement of PD stents to bridge the leak site or stricture. However, because it has not been clearly confirmed that the introduction of stents leads to permanent recovery of PD and permanent cessation of the leakage of pancreatic juice after the stent removal, the use of this intervention is questionable. The reason for this is that it may represent overtreatment in these patients, given the mechanical trauma of the placement and the removal of the PD stent, that the two demanding interventions (ERCP) are performed under conscious sedation, and the cost-benefit effect.

Laparoscopic management has been reported with encouraging results, but long-term follow-up results have yet to show equivalence to open surgery and other minimally invasive methods. The disadvantage of laparoscopic surgery is that it may not be suitable for patients who are unfit to undergo general anaesthesia or for patients with a history of extensive previous abdominal surgery.

Currently, few randomized controlled studies have been performed that compare the various minimally invasive approaches in the management of PPCs. Several groups worldwide have developed new minimally invasive approaches for the treatment of PPC. Applicability of these techniques is highly dependent on the availability of specialized expertise and multidisciplinary teams that are dedicated to the management of pancreatic diseases. This review article is intended to help physicians base their therapeutic decisions about minimally invasive management of PPCs on the current state of therapeutic technology

and published data.

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